Published in final edited form as:

Disabil Health J. 2016 July ; 9(3): 544–551. doi:10.1016/j.dhjo.2016.01.005.

Demographic profile of families and children in the Study to Explore Early Development (SEED): Case-control study of autism spectrum disorder

Carolyn G. DiGuiseppi, M.D., M.P.H., Ph.D.[a,*], **Julie L. Daniels, Ph.D.**[b], **Daniele M. Fallin, Ph.D.**[c], **Steven A. Rosenberg, Ph.D.**[d], **Laura A. Schieve, Ph.D.**[e], **Kathleen C. Thomas, Ph.D.**[f], **Gayle C. Windham, Ph.D.**[g], **Cynthia W. Goss, M.A.**[a,1], **Gnakub N. Soke, M.D., Ph.D.**[a,2], **Dustin W. Currie, M.P.H.**[a], **Alison B. Singer, Ph.D.**[h,3], **Li-Ching Lee, Ph.D.**[h], **Pilar Bernal, Ph.D.**[i,4], **Lisa A. Croen, Ph.D.**[j], **Lisa A. Miller, M.D., M.S.P.H.**[k], **Jennifer A. Pinto-Martin, Ph.D.**[l], **Lisa M. Young, M.P.H.**[l], and **Diana E. Schendel, Ph.D.**[e,5]

[a]Department of Epidemiology, Colorado School of Public Health, Campus Box B119, 13001 E. 17th Place, Aurora, CO 80045, USA

[b]Department of Epidemiology, Gillings School of Global Public Health, University of North Carolina at Chapel Hill, CB #7435, Chapel Hill, NC 27599-7435, USA

[c]Wendy Klag Center for Autism and Developmental Disabilities, Department of Mental Health, Johns Hopkins Bloomberg School of Public Health, 624 N. Broadway, HH 850, Baltimore, MD 21205, USA

[d]Department of Psychiatry, University of Colorado School of Medicine, Campus Box F546, 13001 E. 17th Place, Aurora, CO 80045, USA

[e]National Center on Birth Defects and Developmental Disabilities, Centers for Disease Control and Prevention, Atlanta, GA 30329, USA

[f]Cecil G. Sheps Center for Health Services Research, University of North Carolina at Chapel Hill, 725 Martin Luther King Jr. Blvd., CB#7590, Chapel Hill, NC 27599-7590, USA

[g]California Department of Public Health, 850 Marina Bay Pkwy, Bldg. P/EHIB, Richmond, CA 94804, USA

[h]Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, 615 N Wolfe St, Baltimore, MD 21205, USA

[*] Corresponding author. Tel.: +1 303 724 4415. Carolyn.DiGuiseppi@ucdenver.edu (C.G. DiGuiseppi).

[1]Current address: Centers for American Indian and Alaska Native Health, Campus Box F800, Nighthorse Campbell Native Health Building, 13055 E 17th Avenue, Aurora, CO 80045, USA.

[2]Current address: Centers for Disease Control and Prevention, 1600 Clifton Road NE, Mailstop E-86, Atlanta, GA 30329-4027, USA.

[3]Current address: Department of Epidemiology, CB #7435, University of North Carolina Gillings School of Global Public Health, Chapel Hill, NC 27599-7435, USA.

[4]Current address: ASPIRE Program, El Camino Hospital, Behavioral Health Services, 2500 Grant Rd, Mountain View, CA 94040-4378, USA.

[5]Current address: Department of Public Health, Institute of Epidemiology and Social Medicine, Aarhus University, Bartholins Allé 2, Bldg 1260, DK-8000 Aarhus C, Denmark.

Conflict of interest statement: None of the authors report any conflicts of interest.

[i]Kaiser Permanente, ASD Center, Department of Psychiatry, 6620 Via del Oro, San Jose, CA 95119, USA

[j]Autism Research Program, Kaiser Permanente Division of Research, 2000 Broadway, Oakland, CA 94612, USA

[k]Division of Disease Control and Environmental Epidemiology, Colorado Department of Public Health and Environment, 4300 Cherry Creek Drive South, Denver, CO 80246-1530, USA

[l]Center for Autism and Developmental Disabilities, Research and Epidemiology (CADDRE), University of Pennsylvania, School of Nursing, 418 Curie Blvd, Claire Fagan Hall, Philadelphia, PA 19104, USA

Abstract

Background—The Study to Explore Early Development (SEED) is designed to enhance knowledge of autism spectrum disorder characteristics and etiologies.

Objective—This paper describes the demographic profile of enrolled families and examines sociodemographic differences between children with autism spectrum disorder and children with other developmental problems or who are typically developing.

Methods—This multi-site case-control study used health, education, and birth certificate records to identify and enroll children aged 2–5 years into one of three groups: 1) cases (children with autism spectrum disorder), 2) developmental delay or disorder controls, or 3) general population controls. Study group classification was based on sampling source, prior diagnoses, and study screening tests and developmental evaluations. The child's primary caregiver provided demographic characteristics through a telephone (or occasionally face-to-face) interview. Groups were compared using ANOVA, chi-squared test, or multinomial logistic regression as appropriate.

Results—Of 2768 study children, sizeable proportions were born to mothers of non-White race (31.7%), Hispanic ethnicity (11.4%), and foreign birth (17.6%); 33.0% of households had incomes below the US median. The autism spectrum disorder and population control groups differed significantly on nearly all sociodemographic parameters. In contrast, the autism spectrum disorder and developmental delay or disorder groups had generally similar sociodemographic characteristics.

Conclusions—SEED enrolled a sociodemographically diverse sample, which will allow further, in-depth exploration of sociodemographic differences between study groups and provide novel opportunities to explore sociodemographic influences on etiologic risk factor associations with autism spectrum disorder and phenotypic subtypes.

Keywords

Autism spectrum disorder; Epidemiology; Socioeconomic factors; Demographics

Estimates of the prevalence of autism spectrum disorder (ASD) have increased dramatically since autism was first described,[1,2] but understanding of ASD etiology and risk factors remains limited (reviewed in Ref. 3). Although the etiologic complexity of ASDs and the likely causal interplay among genetic, epigenetic, environmental, and lifestyle risk factors

has been recognized,[4–9] relatively little is known about how potentially modifiable exogenous factors affect ASD risk. Studies have often used small clinical samples in which inferences have been limited by use of clinic-based controls, low power, or lack of data on covariates that may affect exposure-disease relationships. Population-based studies using administrative data or health registries have identified associations between ASD risk and parental age,[10,11] and prenatal and perinatal suboptimality,[12,13] but often lack detail with regard to ASD characterization, genetics, and non-genetic risk factors important in etiologic investigations.[14–16] While several large, epidemiologic studies are examining environmental exposures among phenotypically well-characterized children with ASD,[17,18] a more complete understanding of the epidemiology and complex etiology of ASD requires investigations that combine detailed ASD phenotypic characterization, appropriate comparison groups, genetic data, and relevant data on modifiable risk factors in large, geographically diverse samples.

In response to this need, the Study to Explore Early Development (SEED), a case-control study designed to examine etiologic risk factors for ASD in young children and their families, was launched. SEED's design incorporates several unique features that strengthen its ability to answer important questions about ASD.[19] SEED includes two comparison groups—children without ASD randomly sampled from the birth population and children with non-ASD neurodevelopmental disorders—to control for recall bias and more accurately characterize features specific to ASD. This paper's purpose is to describe the sociodemographic characteristics of the SEED sample and to conduct a preliminary exploration of differences in these characteristics between children with ASD and children in the two comparison groups.

Methods

SEED is a multi-site, case-control study, the methods for which have been described previously.[19] Cases (ASD group) comprise children with ASD verified by clinical evaluation. The two comparison groups include a general Population group and a Developmental Delays/Disorders group. Eligible children were born between September 1, 2003 and August 31, 2006 in a study catchment area, resided there at first study contact, and lived with a family member or other caregiver aged ≥18 years, who had resided with and consistently cared for the child since age 6 months and who spoke English (or, at two sites, English or Spanish). Characteristics of the study catchment areas and their birth populations have been described.[19] To maintain the appropriate age range for validated study instruments, children were enrolled so as to be 30.0–68.9 months old at the time of the developmental evaluation.

ASD and Developmental Delays/Disorders group children were ascertained from clinical and educational settings serving or evaluating children with developmental problems, and included children who had received either an ASD or related clinical diagnosis or early intervention or special education services for an ASD or related condition.[19] Related diagnoses and conditions were broadly defined to capture both previously diagnosed and undiag-nosed children with ASD. Population group children were identified by randomly

sampling birth certificates for births during the cohort period to mothers who were resident in the study catchment area.

After an introductory letter, families were screened for eligibility by telephone and, if eligible, were administered the Social Communication Questionnaire (SCQ).[20] The SCQ score was used to identify previously undiagnosed children at risk for ASD based on a score of 11 or higher.[21,22]

Uniform data were collected in all three study groups by parent-completed interviews, forms and questionnaires and clinical developmental assessments.[19] Children previously diagnosed with ASD, or at risk for ASD based on the SCQ, completed additional clinical developmental assessments.

Methods for assigning final group classification have been detailed elsewhere.[23] Briefly, children with a previous ASD diagnosis, an SCQ score of 11 or higher, or any ASD symptoms observed during administration of the Mullen Scales of Early Learning (MSEL)[24] (which was given to all enrolled children) were also administered the Autism Diagnostic Observation Schedule (ADOS)[25,26] and the Autism Diagnostic Interview Revised (ADI-R).[27–29] Children undergoing the comprehensive evaluation who met ASD cutoff scores were classified as ASD group,[19,23] and otherwise were classified as Developmental Delays/Disorders group with an additional *post-hoc* sub-classification, “with ASD characteristics.” Children with a previously diagnosed developmental condition who had none of the indications (above) for needing a comprehensive ASD evaluation were classified as Developmental Delays/Disorders group with a *post-hoc* sub-classification, “without ASD characteristics.” Children recruited through birth certificate sampling whose initial SCQ score was < 11 comprised the Population group.

The identified caregiver completed a telephone or, rarely, in-person interview about family, child and household characteristics. Respondents who were the biologic mother (99%) additionally provided a reproductive and pregnancy history. The caregiver interview collected maternal and paternal education (highest year of schooling completed, categorized as collected), age at child's birth (years), ethnicity (Hispanic Yes/No), race (American Indian or Alaska Native, Asian, Black or African American, Native Hawaiian or Other Pacific Islander, White), birth country, and age at immigration to the US (years). Respondents were also asked household income (categorized as collected), language usually spoken at home (English, Spanish, or other language), and number of people living in the household and, of these, number aged < 18 years at interview.

Analyses were conducted among children with a final study classification (ASD, Developmental Delays/Disorders or Population group), whose parent or caregiver responded to the interview. Participants with missing data on maternal characteristics (< 2% of children; range .1–1.3% for the characteristics measured), paternal characteristics (< 5%; range 2.2–4.9%), and household characteristics (< 5%; 1.4–4.8%) were excluded from analyses. Descriptive statistics are reported for each study group. Variations among the groups, overall and between group pairs, were examined using multinomial regression. Model fit was assessed using the likelihood-ratio test. The Wald statistic was used to assess

between-group differences for individual predictors in each model. Means were compared using a Welch or ANOVA test, depending on equality of variances. Children classified as Developmental Delays/Disorders with versus without ASD characteristics were compared using chi-square or *t*-tests as appropriate. An alpha of .05 was used.

An in-person developmental assessment was necessary for final classification into the ASD group. Therefore, children scheduled for comprehensive evaluation who did not complete it during their clinic visit ($n = 122$; 10.3%) were excluded from this analysis. In contrast, children with an SCQ score < 11 could be classified into the Developmental Delays/Disorders or Population groups based solely on their ascertainment source (i.e., clinical or educational sources and birth certificate sampling, respectively), without any developmental evaluation. Hence, children in either group with SCQ score < 11 and a caregiver interview but no in-person clinic visit for evaluation ($n = 197$) were retained in analyses. Although the absence of a developmental evaluation could have led to misclassification, fewer than five children are estimated to have been misclassified, extrapolating from similar children who did complete the in-person evaluation. Because of potential sociodemographic differences between families who did and did not complete the in-person visit, comparisons were repeated on a subgroup limited to families who had both a caregiver interview and in-person visit, as a sensitivity analysis.

To examine heterogeneity in self-reported sociodemographic characteristics across sites, logistic regression modeling of ASD versus Population group children was run with interaction terms between site and maternal race, maternal education and total household income.

In order to assess characteristics of mothers of enrolled Population group children versus those of the source population birth cohort, birth certificate data on maternal age, race and education (available from five sites) and ethnicity (from four sites) were compared using chi-square or *t*-tests as appropriate.

This study was approved by Institutional Review Board (IRB)-C, CDC Human Research Protection Office; Kaiser Foundation Research Institute (KFRI) Kaiser Permanente Northern California IRB, Colorado Multiple IRB, Emory University IRB, Georgia Department of Public Health IRB, Maryland Department of Health and Mental Hygiene IRB, Johns Hopkins Bloomberg School of Public Health Review Board, University of North Carolina IRB and Office of Human Research Ethics, IRB of The Children's Hospital of Philadelphia, and IRB of the University of Pennsylvania. The study has been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. All enrolled families provided written consent for participation.

Results

Of 3769 children enrolled between December 2007 and September 2011, the caregivers of 3048 (80.9%) children were interviewed. There were 2768 children whose care-givers were interviewed and who received a final classification, including 697 (25.2%) in the ASD group, 1084 (39.2%) in the Developmental Delays/Disorders group (305 [11.0%] with and

779 [28.1%] without ASD characteristics), and 987 (35.7%) in the Population group. Most caregiver respondents (99.0%) were biological mothers; .6% were biological fathers and .3% other family members. The respondent's relationship to the child did not differ between groups ($p = .35$). Of enrolled children, 31.7% were born to non-White mothers, 11.4% to Hispanic mothers, and 17.6% to foreign-born mothers.

Child sex differed significantly between groups in the expected direction[3] (Table 1). Average child age at enrollment for the entire sample was 55.7 months (range 29.1–68.1 months) and was similar between groups. Compared to parents of Population group children, parents of ASD group children were more likely to be of Black or Asian race, foreign birth, low education and low household income, and speak a language other than English, and mothers of ASD group children were more likely to be of Hispanic ethnicity. Mean maternal and paternal age did not differ between these two groups.

There were fewer sociodemographic differences between the ASD and Developmental Delays/Disorders groups. Mothers of ASD group children were more likely than mothers of Developmental Delays/Disorders group children to be Asian, speak a language other than English and have low education. Fathers of ASD group children were also more likely to be Asian and were older. Maternal age did not differ between these two groups. In sensitivity analyses, results restricted to children who completed the in-person visit did not differ in magnitude, direction or statistical significance from the reported results.

The sociodemographic differences between study groups described above were not driven by any single site, and adjustment for site did not substantively change any of the observed associations.

The two *post-hoc* Developmental Delays/Disorders groups are shown in Table 2. Like ASD group children, Developmental Delays/Disorders group children with ASD characteristics were more likely to have Black or Hispanic mothers, parents whose primary language was not English and who had not completed a college or advanced degree, and lower household income, than those without ASD characteristics.

Compared to mothers of the source population birth cohort, mothers of Population group children from the same sites were significantly older (mean age 31.8 [5.4] vs. 28.9 [6.2]), and less likely to be Black (14.9% vs. 20.1%), Asian (5.1% vs. 10.7%), or Hispanic (11.3% vs. 25.6%), and to have completed 12 or fewer years of education (11.1% vs. 43.3%); $p < .0001$ for all comparisons.

Discussion

Through the use of a broad diagnostic net, involving both clinical and educational recruitment sources, SEED successfully enrolled a highly diverse sample of participants, including minorities and low socioeconomic status families, with distributions comparable to the racial and ethnic diversity in the United States.[30] While a number of large, population-based studies of ASD using surveil-lance or administrative data have been conducted in North America, (e.g. Refs. 1,31–33) SEED improves substantially on existing population studies by providing richer data for analysis coupled with a large, well-defined study sample.

This descriptive study identified a wide range of sociodemographic differences among children with ASD, children with other developmental disabilities, and children from the reference population, which merit deeper exploration. Documented ASD prevalence in the US has been highest among children from families of higher socioeconomic status.[3,9,34] However, it remains difficult in US studies to distinguish sociodemographic characteristics that may affect disease occurrence from factors that may enhance ASD diagnosis, such as access to health care and educational testing.[35] In countries where such barriers are reduced for disadvantaged groups, the association between ASD and socioeconomic status is absent or even reversed, with higher risk in families of lower socioeconomic status,[36–38] similar to the findings in our study. The prevalence of ASD has also been observed to vary by race and ethnicity, with lower rates generally reported among Hispanic or Black non-Hispanic children than among White non-Hispanic children in many studies.[1,9,32,34,39] These apparent differences may be due to differences in socioeconomic status, parental awareness, or cultural sensitivities, although lower rates among blacks and Hispanics have persisted even after adjusting for potentially confounding factors in some studies.[32,39] Under-ascertainment of ASD among minorities is another explanation suggested by analyses of surveillance and other population-based data.[1,35,39] However, several recent studies have reported findings consistent with our results, with higher proportions of ASD among Hispanic and Black non-Hispanic children compared to White non-Hispanic children.[40,41] There is also a growing literature on the relationship between parents' ages at a child's birth and ASD,[10,11] nearly all of which suggests a moderate positive association between advanced maternal or paternal age, or both, and ASD. While several biologic explanations for these findings are plausible and need further study, ascertainment bias and sociodemographic explanations must also be considered. The latter might also explain the lack of an association between advanced parental age and ASD in the current study.

Families enrolled in the Population control group were more likely to be White, non-Hispanic and highly educated than those in the source population. However, the extent to which differential participation in the Population control group by sociodemographic characteristics might bias our results is difficult to assess in the absence of similar information for the ASD or Developmental Delays/Disorders study groups. We excluded 10% of children at risk for ASD because of instrument non-completion (most commonly because the child had low mental abilities and ASD could not be distinguished from intellectual disability or other developmental disorders). Because families of children who did and did not complete the evaluation were similar in nearly all sociodemographic characteristics, this exclusion is unlikely to have substantially biased our results. This report describes how a range of sociodemographic characteristics is distributed between study groups in the SEED sample and is not intended to provide a generalizable assessment of sociodemographic differences between children with and without ASD in the US population.

Conclusion

The primary goal of SEED is to test important hypotheses related to ASD phenotype and etiology, through collection of in-depth data that will enable the testing of multiple different, but potentially interrelated, hypotheses. The current report demonstrates that SEED methods yielded a well-defined and socioeconomically diverse study sample that will provide novel

opportunities to explore the influence of socioeconomic characteristics on etiologic risk factor associations with ASD and ASD phenotypic subtypes. Further, observed differences between study groups merit additional studies with in-depth analyses of SEED data to determine independent associations of diverse sociodemographic factors.

Acknowledgments

Patrick Thompson made important contributions to data selection, management and cleaning for this paper. The investigators gratefully acknowledge the project staff and the enrolled families. A preliminary version of these results was presented at the International Meeting for Autism Research, Toronto, Canada, on May 18, 2012.

Disclosures: This project was supported by Centers for Disease Control and Prevention (CDC) Cooperative Agreements announced under the following RFAs: 01086, 02199, DD11-002, DD06-003, DD04-001, and DD09-002. The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

References

1. Centers for Disease Control and Prevention. Prevalence of autism spectrum disorder among children aged 8 years – Autism and Developmental Disabilities Monitoring Network, 11 sites, United States, 2010. MMWR Surveill Summ. 2014; 63(SS02):1–21.
2. Elsabbagh M, Divan G, Koh YJ, et al. Global prevalence of autism and other pervasive developmental disorders. Autism Res. 2012; 5:160–179. [PubMed: 22495912]
3. Newschaffer CJ, Croen LA, Daniels J, et al. The epidemiology of autism spectrum disorders. Annu Rev Public Health. 2007; 28:235–258. [PubMed: 17367287]
4. Bill BR, Geschwind DH. Genetic advances in autism: heterogeneity and convergence on shared pathways. Curr Opin Genet Dev. 2009; 19:271–278. [PubMed: 19477629]
5. Dufour-Rainfray D, Vourc'h P, Tourlet S, Guilloteau D, Chalon S, Andres CR. Fetal exposure to teratogens: evidence of genes involved in autism. Neurosci Biobehav Rev. 2011; 35:1254–1265. [PubMed: 21195109]
6. Grafodatskaya D, Chung B, Szatmari P, Weksberg R. Autism spectrum disorders and epigenetics. J Am Acad Child Adolesc Psychiatry. 2010; 49:794–809. [PubMed: 20643313]
7. Herbert MR. Contributions of the environment and environmentally vulnerable physiology to autism spectrum disorders. Curr Opin Neurol. 2010; 23:103–110. [PubMed: 20087183]
8. Voineagu I. Gene expression studies in autism: moving from the genome to the transcriptome and beyond. Neurobiol Dis. 2012; 45:69–75. [PubMed: 21839838]
9. Durkin MS, Maenner MJ, Meaney FJ, et al. Socioeconomic inequality in the prevalence of autism spectrum disorder: evidence from a US cross-sectional study. PLoS One. 2010; 5:e11551. [PubMed: 20634960]
10. Hultman CM, Sandin S, Levine SZ, Lichtenstein P, Reichenberg A. Advancing paternal age and risk of autism: new evidence from a population-based study and a meta-analysis of epidemiological studies. Mol Psychiatry. 2011; 16:1203–1212. [PubMed: 21116277]
11. Sandin S, Hultman CM, Kolevzon A, Gross R, MacCabe JH, Reichenberg A. Advancing maternal age is associated with increasing risk for autism: a review and meta-analysis. J Am Acad Child Adolesc Psychiatry. 2012; 51:477–486. [PubMed: 22525954]
12. Gardener H, Spiegelman D, Buka SL. Perinatal and neonatal risk factors for autism: a comprehensive meta-analysis. Pediatrics. 2011; 128:344–355. [PubMed: 21746727]
13. Dodds L, Fell DB, Shea S, Armson BA, Allen AC, Bryson S. The role of prenatal, obstetric and neonatal factors in the development of autism. J Autism Dev Disord. 2011; 41:891–902. [PubMed: 20922473]
14. Becerra TA, Wilhelm M, Olsen J, Cockburn M, Ritz B. Ambient air pollution and autism in Los Angeles County, California. Environ Health Perspect. 2013; 12:380–386. [PubMed: 23249813]

15. Croen LA, Grether JK, Yoshida CK, Odouli R, Hendrick V. Antidepressant use during pregnancy and childhood autism spectrum disorders. Arch Gen Psychiatry. 2011; 68:1104–1112. [PubMed: 21727247]
16. Windham GC, Sumner A, Li SX, et al. Use of birth certificates to examine maternal occupational exposures and autism spectrum disorders in offspring. Autism Res. 2013; 6:57–63. [PubMed: 23361991]
17. Hertz-Picciotto I, Croen LA, Hansen R, Jones CR, Van de Water J, Pessah IN. The CHARGE study: an epidemiologic investigation of genetic and environmental factors contributing to autism. Environ Health Perspect. 2006; 114:1119–1125. [PubMed: 16835068]
18. Stoltenberg C, Schjølberg S, Bresnahan M, et al. The Autism Birth Cohort: a paradigm for gene-environment-timing research. Mol Psychiatry. 2010; 15:676–680. [PubMed: 20571529]
19. Schendel DE, DiGuiseppi C, Croen LA, et al. The Study to Explore Early Development (SEED): a multisite epidemiologic study of autism by the Centers for Autism and Developmental Disabilities Research and Epidemiology (CADDRE) network. J Autism Dev Disord. 2012; 42:2121–2140. [PubMed: 22350336]
20. Rutter, ML.; Bailey, A.; Lord, C. The Social Communication Questionnaire: Manual. Western Psychological Services; 2003.
21. Allen CW, Silove N, Williams K, Hutchins P. Validity of the social communication questionnaire in assessing risk of autism in preschool children with developmental problems. J Autism Dev Disord. 2007; 37:1272–1278. [PubMed: 17080270]
22. Lee LC, David AB, Rusyniak J, Landa R, Newschaffer CJ. Performance of the Social Communication Questionnaire in children receiving preschool special education services. Res Autism Spectr Disord. 2007; 1:126–138.
23. Wiggins LD, Reynolds A, Rice CE, et al. Using standardized diagnostic instruments to classify children with autism in the Study to Explore Early Development. J Autism Dev Disord. 2015; 45:1271–1280. PMID: 25348175. [PubMed: 25348175]
24. Mullen, E. Mullen Scales of Early Learning. AGS. , editor. American Guidance Service, Inc; Circle Pines, MN: 1995.
25. Lord, C.; Rutter, M.; DiLavore, PC.; Risi, S. Autism Diagnostic Observation Schedule-WPS (ADOS-WPS). Western Psychological Services; Los Angeles, CA: 1999.
26. Gotham K, Risi S, Pickles A, Lord C. The Autism Diagnostic Observation Schedule: revised algorithms for improved diagnostic validity. J Autism Dev Disord. 2007; 37:613–627. [PubMed: 17180459]
27. Rutter, M.; Le Couteur, A.; Lord, C. Manual. Western Psychological Services; Los Angeles, CA: 2003. The Autism Diagnostic Interview Revised..
28. Le Couteur A, Rutter M, Lord C, et al. Autism diagnostic interview: a semi-structured interview for parents and caregivers of autistic persons. J Autism Dev Disord. 1989; 19:363–387. [PubMed: 2793783]
29. Risi S, Lord C, Gotham K, et al. Combining information from multiple sources in the diagnosis of autism spectrum disorders. J Am Acad Child Adolesc Psychiatry. 2006; 45:1094–1103. [PubMed: 16926617]
30. Humes, KR.; Jones, NA.; Ramirez, RR. Overview of Race and Hispanic Origin: 2010. 2010 Census Briefs.; U.S. Census Bureau: 2011. Available, http://www.census.gov/prod/cen2010/briefs/c2010br-02.pdf [29.12.15]
31. Bertrand J, Mars A, Boyle C, Bove F, Yeargin-Allsopp M, Decoufle P. Prevalence of autism in a United States population: the Brick Township, New Jersey, investigation. Pediatrics. 2001; 108:1155–1161. [PubMed: 11694696]
32. Kogan MD, Blumberg SJ, Schieve LA, et al. Prevalence of parent-reported diagnosis of autism spectrum disorder among children in the US, 2007. Pediatrics. 2009; 124:1395–1403. [PubMed: 19805460]
33. Grether JK, Anderson MC, Croen LA, Smith D, Windham GC. Risk of autism and increasing maternal and paternal age in a large North American population. Am J Epidemiol. 2009; 170:1118–1126. [PubMed: 19783586]

34. Windham GC, Anderson MC, Croen LA, Smith KS, Collins J, Grether JK. Birth prevalence of autism spectrum disorders in the San Francisco Bay area by demographic and ascertainment source characteristics. J Autism Dev Disord. 2011; 41:1362–1372. [PubMed: 21264681]

35. Mandell DS, Wiggins LD, Carpenter LA, et al. Racial/ethnic disparities in the identification of children with autism spectrum disorders. Am J Public Health. 2009; 99:493–498. [PubMed: 19106426]

36. Campbell, M.; Reynolds, L.; Cunningham, J.; Minnis, H.; Gillberg, CG. Autism in Glasgow: cumulative incidence and the effects of referral age, deprivation and geographical location.. Child Care Health Dev. 2011. http://dx.doi.org/10.1111/j.1365-2214.2011.01340.x

37. Larsson HJ, Eaton WW, Madsen KM, et al. Risk factors for autism: perinatal factors, parental psychiatric history, and socioeconomic status. Am J Epidemiol. 2005; 161:916–925. [PubMed: 15870155]

38. Rai D, Lewis G, Lundberg M, et al. Parental socioeconomic status and risk of offspring autism spectrum disorders in a Swedish population-based study. J Am Acad Child Adolesc Psychiatry. 2012; 51:467–476. [PubMed: 22525953]

39. Palmer RF, Walker T, Mandell DS, Bayles B, Miller CS. Explaining low rates of autism among Hispanic schoolchildren in Texas. Am J Public Health. 2010; 100:270–272. [PubMed: 20019320]

40. Becerra TA, von Ehrenstein OS, Heck JE, Olsen J, Arah OA, Jeste SS. Autism spectrum disorders and race, ethnicity, and nativity: a population-based study. Pediatrics. 2014; 134:e63–e71. [PubMed: 24958588]

41. Keen DV, Reid FD, Arnone D. Autism, ethnicity and maternal immigration. Br J Psychiatry. 2010; 196:274–281. [PubMed: 20357302]

Table 1

Distribution of sociodemographic characteristics among SEED participant groups: children with Autism Spectrum Disorders (ASD) or Developmental Disabilities (DD) and Population Controls (POP)

Sociodemographic characteristics	ASD (*N* = 697) %(*N*)	DD (*N* = 1084) % (*N*)	POP (*N* = 987) % (*N*)	*p*-value[a]
Child sex — male	81.8 (570)[†,‡]	66.1 (717)[§]	54.0 (533)	<.0001
Child year of birth				< .0001
2003	8.8 (61)[†,‡]	5.2 (56)	5.7 (56)	
2004	31.1 (217)	34.1 (370)	39.9 (394)	
2005 (ref)	39.4 (275)	44.8 (486)	45.4 (448)	
2006	20.7 (144)[†,‡]	15.9 (172)[§]	9.0 (89)	
Mother's race				< .0001
White (ref)	62.5 (433)	65.9 (702)	75.0 (731)	
Black	20.1 (139)[†]	19.2 (205)[§]	13.2 (129)	
Asian	8.7 (60)[†,‡]	4.4 (47)	4.4 (43)	
Native American/Hawaiian/Pacific Islander	.7 (5)	.7 (7)	.7 (7)	
Hispanic — race not specified	4.0 (28)[†]	5.1 (54)[§]	2.4 (24)	
Multiple races	4.0 (28)	4.7 (50)	4.2 (41)	
Mother's ethnicity — Hispanic	12.2 (85)[†]	13.6 (146)[§]	8.4 (83)	.001
Mother foreign born	21.8 (152)[†]	18.3 (196)[§]	13.7 (135)	< .0001
Mother's primary language				< .0001
English (ref)	88.5 (616)	87.7 (941)	93.3 (918)	
Spanish	5.0 (35)[†,‡]	8.2 (88)[§]	2.8 (28)	
Other	6.5 (45)[†,‡]	4.1 (44)	3.8 (38)	
Mother's highest education				< .0001
Less than high school	5.0 (35)[†,‡]	8.0 (86)[§]	3.1 (31)	
High school	11.4 (79)[†]	11.6 (124)[§]	7.2 (71)	
Some college/trade	31.5 (219)[†]	26.6 (286)[§]	24.6 (242)	
Bachelor's degree (ref)	30.6 (212)	30.1 (323)	35.3 (347)	
Advanced degree	21.5 (149)	23.7 (254)	29.8 (292)	
Father's race				.0002
White (ref)	63.2 (426)	66.5 (696)	73.5 (706)	
Black	21.8 (147)[†]	20.1 (210)[§]	15.7 (151)	
Asian	7.4 (50)[†,‡]	4.3 (45)	3.9 (38)	
Native American/Hawaiian/Pacific Islander	.6 (<.5)	.5 (5)	.9 (9)	
Hispanic — race not specified	4.5 (30)	5.4 (57)[§]	3.3 (32)	
Multiple races	2.5 (17)	3.2 (34)	2.5 (24)	

Sociodemographic characteristics	ASD (*N* = 697) %(*N*)	DD (*N* = 1084) % (*N*)	POP (*N* = 987) % (*N*)	*p*-value[a]
Father's ethnicity — Hispanic	11.5 (78)	13.5 (142)[§]	9.3 (90)	.014
Father foreign born	19.6 (133)[†]	18.8 (199)[§]	14.0 (136)	.004
Father's primary language				< .0001
English (ref)	88.2 (593)	87.5 (921)	92.7 (891)	
Spanish	5.9 (40)[†]	8.3 (88)[§]	3.4 (33)	
Other	5.9 (40)[†]	4.2 (44)	3.9 (37)	
Father's highest education				< .0001
Less than high school	7.4 (50)[†]	9.9 (102)[§]	4.1 (39)	
High school	19.9 (134)[†]	19.4 (200)	14.5 (138)	
Some college/trade	22.2 (149)	22.3 (230)[§]	19.9 (190)	
Bachelor's degree (ref)	28.3 (190)	28.4 (293)	33.2 (317)	
Advanced degree	22.2 (149)	20.0 (207)	28.3 (270)	
Household income in past 12 months				< .0001
<$10,000	7.4 (50)[†]	9.4 (94)[§]	5.5 (52)	
$10,000—30,000	17.5 (119)[†]	14.1 (143)[§]	8.7 (82)	
$30,000—50,000	11.9 (81)[†]	14.2 (144)[§]	11.1 (105)	
$50,000—70,000	14.3 (97)[†]	12.9 (131)[§]	11.6 (110)	
$70,000—90,000	12.9 (88)	13.2 (134)[§]	14.2 (134)	
$90,000—110,000	11.6 (79)	12.1 (123)[§]	13.3 (126)	
>$110,000 (ref)	24.2 (164)	24.0 (243)	35.5 (336)	
Enrollment site distribution				.016
California (ref)	15.5 (108)	14.6 (158)	15.5 (153)	
Colorado	20.2 (141)	16.8 (182)	20.4 (201)	
Georgia	19.6 (137)	22.6 (245)	18.3 (181)	
Maryland	15.5 (108)	12.3 (133)	14.9 (147)	
North Carolina	14.8 (103)[‡]	20.5 (222)	17.4 (172)	
Pennsylvania	14.3 (100)	13.3 (144)	13.5 (133)	

	Mean (SD)	Mean (SD)	Mean (SD)	
Child age at enrollment (months)	55.6 (6.8)	55.8 (7.4)	55.5 (7.6)	.660
Mother's age at child's birth (years)	31.6 (5.5)	31.5 (5.8)	31.9 (5.4)	.394
Father's age at child's birth (years)	34.5 (6.7)[‡]	33.7 (6.4)	34.1 (6.1)	.039
Mother's # years in US at child's birth (if foreign born) (years)	11.9 (9.2)	10.8 (9.0)[§]	14.0 (11.0)	.022
Father's # years in US at child's birth (if foreign born) (years)	14.5 (9.0)	12.5 (9.5)	13.9 (10.2)	.174
Current number living in household	4.3 (1.2)	4.3 (1.3)	4.4 (1.1)	.630
Current number aged < 18 yrs living in household	2.2 (.9)	2.3 (1.0)	2.3 (.9)	.138

[a] *p*-values indicate overall variation among the groups for each factor, examined using multinomial regression. Symbols indicate significant between-group differences for individual predictors in each model

[†] ASD vs. POP $p < .05$

[‡] ASD vs. DD $p < .05$

[§] DD vs. POP $p < .05$.

Table 2

Distribution of sociodemographic factors among SEED participants with developmental disabilities (DD), with and without symptoms of autism spectrum disorder (ASD)

Sociodemographic characteristics	DD with ASD symptoms (*N* = 305) % (*N*)	DD without ASD symptoms (*N* = 779) % (*N*)	*p*-value[a]
Child sex — male	75.4 (230)	62.6 (487)	<.0001
Child year of birth			.027
2003	4.6 (14)	5.4 (42)	
2004	28.5 (87)	36.3 (283)	
2005 (ref)	46.9 (143)	44.0 (343)	
2006	20.0 (61)	14.2 (111)	
Mother's race			< .0001
White (ref)	50.5 (154)	72.1 (548)	
Black	31.8 (97)	14.2 (108)	
Asian	3.3 (10)	4.9 (37)	
Native American/Hawaiian/Pacific Islander	.3 (1)	.8 (6)	
Hispanic — race not specified	8.2 (25)	3.8 (29)	
Multiple races	5.9 (18)	4.2 (32)	
Mother's ethnicity Hispanic	17.4 (53)	12.1 (93)	.022
Mother foreign born	21.3 (65)	17.1 (131)	.104
Mother's primary Language			.006
English (ref)	83.6 (255)	89.3 (686)	
Spanish	12.5 (38)	6.5 (50)	
Other	3.9 (12)	4.2 (32)	
Mother's highest education			< .0001
Less than high school	13.7 (42)	5.7 (44)	
High school	17.1 (52)	9.4 (72)	
Some college/trade	35.1 (107)	23.3 (179)	
Bachelor's degree (ref)	20.6 (63)	33.8 (260)	
Advanced degree	13.4 (41)	27.7 (213)	
Father's race			< .0001
White (ref)	52.4 (154)	71.9 (542)	
Black	32.3 (95)	15.3 (115)	
Asian	2.0 (6)	5.2 (39)	
Native American/Hawaiian/Pacific Islander	.0 (0)	.7 (5)	
Hispanic — race not specified	8.5 (25)	4.2 (32)	
Multiple races	4.8 (14)	2.7 (20)	
Father's ethnicity Hispanic	18.2 (54)	11.6 (88)	.004
Father foreign born	22.5 (67)	17.4 (132)	.058
Father's primary language			.0005
English (ref)	81.6 (243)	89.8 (678)	
Spanish	13.4 (40)	6.3 (48)	

Sociodemographic characteristics	DD with ASD symptoms (*N* = 305) % (*N*)	DD without ASD symptoms (*N* = 779) % (*N*)	*p*-value[a]
Other	5.0 (15)	3.8 (29)	
Father's highest education			< .0001
Less than high school	16.6 (47)	7.3 (55)	
High school	27.6 (78)	16.3 (122)	
Some college/trade	24.5 (69)	21.5 (161)	
Bachelor's degree (ref)	18.4 (52)	32.1 (241)	
Advanced degree	12.7 (36)	22.8 (171)	
Household income in past 12 months			< .0001
<$10,000	18.7 (53)	5.6 (41)	
$10,000—30,000	22.9 (65)	10.7 (78)	
$30,000—50,000	17.6 (50)	12.9 (94)	
$50,000—70,000	12.7 (36)	13.0 (95)	
$70,000—90,000	6.0 (17)	16.0 (117)	
$90,000—110,000	7.0 (20)	14.1 (103)	
>$110,000 (ref)	14.8 (42)	27.6 (201)	
Enrollment site distribution			.0007
California (ref)	11.1 (34)	15.9 (124)	
Colorado	16.4 (50)	16.9 (132)	
Georgia	25.9 (79)	21.3 (166)	
Maryland	7.5 (23)	14.1 (110)	
North Carolina	20.6 (63)	20.4 (159)	
Pennsylvania	18.3 (56)	11.3 (88)	

	Mean (SD)	Mean (SD)	
Child age at enrollment (months)	56.3 (7.4)	55.7 (7.4)	.226
Mother's age at child's birth (years)	29.9 (6.2)	32.1 (5.5)	< .0001
Father's age at child's birth (years)	32.3 (6.9)	34.2 (6.2)	< .0001
Mother's # years in US at child's birth (if foreign born) (years)	9.4 (7.9)	11.5 (9.5)	.130
Father's # years in US at child's birth (if foreign born) (years)	9.9 (6.8)	13.8 (10.4)	.002
Current number living in household	4.3 (1.4)	4.3 (1.2)	.671
Current number aged < 18 yrs living in household	2.3 (1.1)	2.3 (1.0)	.966

[a] *p*-values indicate overall variation among the groups for each factor, examined using multinomial regression.